Central Positional Vertigo —Clinico-anatomic Study—

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Abstract

The authors dealt with three cases of central positional vertigo each caused by a different pathogenesis and presented precise autopsy findings in two of the cases after making serial sections of the brainstem and cerebellum. A definite correlation between lesions of the juxta-restiform body and central positional vertigo is suggested, with discussion on the mechanism of central positional vertigo from the clinico-anatomic point of view.

Key words: positional vertigo, juxta-restiform body, nystagmus, vestibulocerebellar pathway

Introduction

Paroxysmal positional vertigo is characterized by the occurrence of paroxysmal vertigo with the head assuming certain critical positions.^{16, 23, 26, 28)} This sort of vertigo can be divided into two quite different types: one is peripheral or benign paroxysmal positioning vertigo,^{8, 12, 28)} the other is central or malignant persistent positional vertigo which was first reported by Bruns (1902)³⁾ in an autopsy report of cysticercosis of the fourth ventricle and is also known as syndrome of Bruns.²⁴⁾ A patient of the former type usual has a lesion or disturbance in the inner ear,^{8, 12)} the latter type in the brain,^{13-15, 17, 28)} although the exact localization of this lesion has not yet been established. For both types, the positional or positioning vertigo appears accompanied by nystagmus in almost all cases.^{7, 19, 22)} Benign positioning nystagmus is found on positioning the head

such that the affected ear is lowermost and the direction of the nystagmus is toward that ear. There is no nystagmus when positioning the head such that the unaffected ear is dependent. However, it is not certain whether malignant or central positional vertigo appears accompanied by as uniform a nystagmus as that observed in benign positional vertigo.

The authors would like to present three cases, with precise autopsy findings of two of the cases, of central positional vertigo in which fixation of the head was interfered with because of an intracerebral destruction of connections between the cerebellum and the vestibular system as well as between the cerebellum and the muscular propriocepting system from the neck. Discussion is made on the mechanism of this type of vertigo on a morphological basis.

Methods

For the autopsy cases (Cases 1 and 3), the brainstem and the cerebellum were mounted in celloidin as one

block after fixation of the brain in 10% formalin, washing in running water, and dehydration with alcohol. The celloidin blocks were cut transversely in 40 μ m thick slices to make complete serial sections. After numbering all sections, every 5th section in Case 1 was stained by luxol fast blue and cresyl violet (LFB-CV, Klüver-Barrera's method), but in Case 3 every 5th section was alternatively stained by Kultschitzky's modification for myelin and LFB-CV. Several other sections from different levels were stained by PTAH, HE, Kultschitzky's method, *etc.* for each of the two cases.

Case Reports

Case 1: The patient, a 75-year-old man, noticed partial facial palsy on the left side 19 months before admission. A tumor in the left parotid gland was surgically removed, which histologically appeared to be an adenocarcinoma. The patient received postoperative radiation. Eight months before admission, an X-ray examination revealed metastatic lesions in the left lung. Two months before admission, he started complaining of vertigo only when and immediately after he turned his head to the right. It persisted without stopping or decreasing in degree while he kept his head in the same position as when the vertigo started. This condition was thought to be due to a metastatic lesion in some part of the brain, but the exact localization could not be confirmed clinically, as it was before the invention of computerized tomography.

On admission, he exhibited remarkable emaciation, left partial facial palsy, and positional vertigo. No nystagmus was observed. His mental and physical condition was quite normal. Two weeks later, he started complaining of occipital headache with oscillopsia. He had to keep his head excessively turned to the left as even slight turning toward the right caused immediately severe vertigo accompanied by jerky horizontal nystagmus to the left that would not stop or decrease in degree unless the position of the head was changed. His condition gradually deteriorated, and he died.

Autopsy findings: The autopsy examination showed bilateral pulmonary metastases and macroscopically normal cerebral hemispheres. The brainstem and the cerebellum were examined after making serial sections. Metastatic lesions in the brain involved the left juxta-restiform body (JRB) and cerebellar tonsil (Fig. 1). Secondary changes were observed in other structures as follows. The bilateral cerebellar cores, especially on the left, were pale in the myelin-stained sections, but the axons were rather well preserved in the silver-stained sections. The left vestibular nerve root was also par-

Fig. 1 Case 1. Transverse section (seen from below) through the juxta-restiform body occupied by a metastatic carcinoma on the left side.

tially degenerated. Many Purkinje cells in the uvula and a number of neurons in the bilateral nucleus fastigii, the left vestibular nuclei, and accessory cuneate nucleus showed retrograde degenerations such as central chromatolysis and pyknosis (Figs. 2 and 3).

Case 2: The patient, a 61-year-old man, suddenly developed severe vertigo. For the first two weeks he kept his head excessively turned to the left and showed clockwise rotatory nystagmus. During that period, active or passive moving of the head immediately caused him severe vertigo and oscillopsia. It was a typical case of central positional vertigo. Plain CTs showed a small high-density area on the right JRB (Fig. 4). His condition was diagnosed as cerebellar hemorrhage with fourth ventricular rupture possibly from an arteriovenous malformation. This, however, could not be detected by vertebral angiography. Ten days later, clockwise rotatory nystagmus was replaced by down beat nystagmus on gazing in every direction. One year later, he showed persistent positional vertigo to some degree when turning his head to either side, rebound nystagmus, down beat nystagmus when looking up, and cerebellar signs (decomposition, hypermetria, dysdiadochokinesis, *etc.*) on the right side. For several months, he had to keep his head in a fixed position with his hand while walking to avoid producing a positional vertigo. Now, two and a half years later, his positional vertigo is no longer characteristic, apart from cerebellar signs on the right side and positioning down beat nystagmus when lying down or getting up. However, he always refrains from turning his head quickly, from continuous stimulation of the head while having a shower, and from washing his face while standing.

Case 3: The patient, a 58-year-old woman, started to feel vertigo with oscillopsia only when she looked toward her right one month before admission. She was also noticed to lean toward her right while walking.

Fig. 2 Case 1. Left inferior vestibular nucleus with most neurons showing pyknosis. LFB-CV stain, $\times 50$.

Fig. 3 Case 1. Left accessory cuneate nucleus with most neurons showing central chromatolysis or pyknosis. LFB-CV stain, $\times 176$, and $\times 440$ for inset.

Fig. 4 Case 2. Plain CT with small high-density area close to right lateral wall of the fourth ventricle (in which the hematoma ruptured).

Three weeks later she complained of continuous severe vertigo and vomiting. An otorhinolaryngologist (one of the authors) noticed positional and positioning jerky nystagmus to the right with horizontal and rotatory components (Fig. 5A). She did not show gaze nystagmus excepting a few beats at the beginning. Normal caloric responses were obtained on both sides.

She was admitted to a hospital because of deterioration of positional vertigo. Three days later she suddenly became deaf on the right side. Her nystagmus also changed to the left from that time on; when she looked forward, upward, downward, and toward her left, she showed coarse gaze nystagmus to the left (Fig. 5B). Eleven days later, she showed facial and corneal analgesia on the right side, loss of taste on the right half

Fig. 5 Case 3. Nystagmograms. A: March 4, vertigo, B: March 11, right deafness, C: March 22, right facial numbness and hemiagusia, D: March 24, right facial palsy and conscious disturbance. a: Gaze nystagmus, b: positional nystagmus with Frenzel's glasses when patient looks forward and turns head to either side, in lying position (*lower row*), and with head hanging at 45° below the horizontal (*upper row*), c: positioning nystagmus with Frenzel's glasses when patient is lying down (*upper square*) and is getting up from supine position (*lower square*). See text for details. $\bigcirc$: no nystagmus, $\ominus$: presence of doubtful nystagmus, $\rightarrow$: nystagmus to the left, $\leftarrow$: nystagmus to the right, $\downarrow$: down beat nystagmus, $\curvearrowright$: with rotatory components, $\Rightarrow$: high frequency nystagmus, $\Rightarrow$: high amplitude nystagmus, $\oplus$: nystagmus to the left with few movements.

of the tongue, right cerebellar signs, and right hemifacial palsy. Her horizontal nystagmus was accompanied by some rotatory components. She also showed positional nystagmus to the left through Frenzel's glasses when she was looking forward or when her head was passively turned to either side (Fig. 5C). CT examination detected a mass lesion in the right cerebello-pontine angle (Fig. 6) with dilated lateral ventricles. Finally, her nystagmus once again changed; the rotatory components disappeared and the amplitude of the positional nystagmus increased although remaining in the same direction except for right turning of the head (Fig. 5D). Her condition deteriorated and she died about two months after the onset of the disease.

Autopsy findings: Autopsy examination revealed that in the medullo-ponto-cerebellar junction on the right was a tumor which was pathohistologically diagnosed as reticulum cell sarcoma-microglioma (malignant lymphoma). The brain showed bilateral tonsillar and upward tentorial herniations. The tumor occupied the following structures on the right side; cerebellar tonsil, middle and inferior cerebellar peduncles, cerebellar medullary core, JRB, vestibular and cochlear nuclei (Fig. 7). Secondary microscopical changes consisted of degeneration of nerve fibers in the right solitary and trigeminal spinal tracts, and of nerve cell changes such as pyknosis, central chromatolysis and decrease in number in the right facial nucleus, nuclei of the spinal trigeminal and solitary tracts, accessory cuneate nucleus, and the contralateral pontine nuclei.

Fig. 6 Case 3. CT with contrast enhancement showing a mass lesion at the right cerebello-ponto-medullary junction.

Discussion

I. Sites of lesions causing central positional vertigo

All of the cases presented, which all showed central positional vertigo during clinical observations, were revealed to have a lesion involving at least the JRB on one side by autopsy examinations with serial sections of the brain (Cases 1 and 3) or by CT examination (Case 2). From the clinico-anatomic analysis, the lesions causing central vertigo involved at least one of the

Fig. 7 Case 3. Transverse section (seen from below) through the ponto-medullary junction, with a right-sided tumor mass occupying the inferior and middle cerebellar peduncles, juxta-restiform body, cerebellar medullary core, cerebellar tonsil, vestibular, and cochlear nuclei, etc.

following possible structures: 1) cerebellar vermis, 2) nucleus fastigii, 3) nuclei vestibulares, 4) JRB, and 5) intracerebral vestibular nerve root. Among them, the authors consider nuclei vestibulares and JRB the most probable sites of the primary lesions responsible for central positional vertigo. Lesions involving the cerebellar vermis or nucleus fastigii may cause vertigo, but it is improbable that the vertigo due to such lesions would appear in relation to a particular head turn. Lesions involving the intracerebral vestibular nerve root may also produce vertigo, but only peripheral-type vertigo, and their occurrence is in any case most improbable. However, a lesion involving the JRB on one side has a greater chance of being connected with head turning than a lesion involving nuclei vestibulares, as our clinico-anatomic observations showed. Why a JRB lesion can be connected with head turning is discussed below.

II. The JRB and its fiber connections

According to anatomic contributions by experimental works,^{1,2,4-6,9,11,18,29)} the JRB is considered to be a bundle of nerve fibers mainly connecting the vestibular nuclei and the cerebellum, *i.e.*, a set of secondary vestibular tracts, and to contain several different nervous pathways (Fig. 8) as follows: 1) the vestibulo-cerebellar pathway which includes the vestibulo-fastigial and vestibulo-vermian pathways (nerve fibers which derive mainly from the inferior vestibular nucleus and partially from the medial nucleus terminate in the homolateral fastigial nucleus, the vermis, and partially in the contralateral fastigial nucleus.)^{2,4,5,9)}, 2) the cerebello-vestibular pathway, *i.e.*, the fastigio-vestibular pathway excluding the uncinata bundle of

Russell (nerve fibers which derive from the fastigial nucleus terminate homolaterally—though some fibers terminate contralaterally—in the vestibular nuclei, especially in the lateral and inferior nuclei.)^{4-6,29)}, 3) the direct homolateral pathway from the vestibular nerve root to the cerebellum including the nodulus, uvula, flocculus, and fastigial nucleus^{2,9,29)}, 4) the cuneo-cerebellar pathway (the external or accessory cuneate nucleus of von Monakow sends fibers to the cerebellum including the anterior lobe, pyramis, nodulus, and uvula.)^{1,10)}, and 5) several others (for example, the reticulo-cerebellar pathway).

Similar results regarding the vestibulo- and cuneo-cerebellar systems have been reported using the horse-radish peroxidase technique.^{20,21,25,27,30)}

III. Secondary histologic changes related to JRB lesions

The above findings on JRB fiber connections — which had been brought to light only in experimental work^{1,2,4-6,9-11,18,29)}—have been here confirmed as applying to the human brain by our findings on histological changes in the brainstem and the cerebel-

Fig. 8 The juxta-restiform body and its fiber connections. VI: abducens nucleus, CR: corpus restiforme (inferior cerebellar peduncle), I: inferior vestibular nucleus, JRB: juxta-restiform body, L: lateral vestibular nucleus, LL: lateral lemniscus, LM: medial lemniscus, N VIII: statocoustic nerve, NCA: accessory cuneate nucleus, ND: dentate nucleus, NE: emboliform nucleus, NF: fastigial nucleus, NG: globosus nucleus, NVE: vestibular nuclei, PCM: middle cerebellar peduncle, TPM: pyramidal tract, TTC: central tegmental tract, VC: cerebellar vermis, VE IV: fourth ventricle.

lum in the two autopsy cases. The most significant changes were: 1) retrograde degeneration in the homolateral vestibular nuclei and accessory cuneate nucleus (Case 1); 2) retrograde degeneration in the homolateral nucleus fastigii, and partially in the contralateral nucleus fastigii (Cases 1 and 3); and 3) partial degeneration in the homolateral vestibular nerve root (Case 1).

These secondary changes are considered to be related to JRB lesions on one side.

IV. Mechanism causing central positional vertigo

The most important question is what is the mechanism of central persistent positional vertigo. The authors believe that the cerebellar vermis could act as a kind of "shock-absorber" to vestibular stimuli under normal conditions. It is considered that the vermis always adjusts usual imbalances between vestibular stimuli and proprioceptive stimuli regarding head or upper trunk movements, or between their right and left functions. Then, a one-sided lesion with sudden onset involving the JRB might cause a disturbance in the balance of labyrinthine stimuli from both internal ears, and at the same time could interfere with neck fixation due to an involvement of the cuneo-cerebellar pathway as the accessory cuneate nucleus relays afferent muscular proprioception fibers from the homolateral cervical and upper thoracic spinal ganglia.^{1,6,10,18)}

In regard to the correlation between the position of the head that causes central positional vertigo and the side of the lesion, there were some discrepancies between Case 1 and the other two cases. The authors have no appropriate explanation for this. The position of the head causing central positional vertigo, however, was similar to that causing peripheral type vertigo at the initial stage in Cases 2 and 3, in which the positional vertigo was observed to occur on turning the head to the side of the lesion.

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